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STRESS FRACTURES: THE REMODELLING RESPONSE TO EXCESSIVE 1/1
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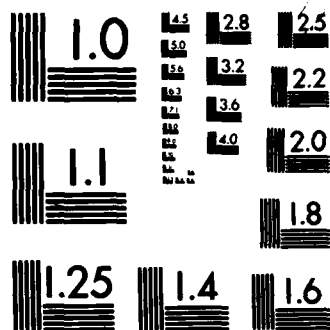
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STRESS FRACTURES: THE REMODELLING RESPONSE TO
EXCESSIVE REPETITIVE LOADING

C.T. Rubin, J. McA. Harris, B.H. Jones, H.B. Ernst, & L.E. Lanyon
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Title, Stress Fractures: The Remodelling Response to Excessive Repetitive Loading

Author(s) C.T. Rubin, J. McA. Harris, B.H. Jones, H.B. Ernst, & L.E. Lanyon
Musculo-Skeletal Research Group of Tufts University

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Location Atlanta, GA

Date February 1984

2. Budget Project No. _____ Cost Code 34581101029

3. Attached contains no classified material. It meets accepted standards for scientific accuracy and propriety. It contains no potentially sensitive or controversial items.



JAMES A. VOGEL, Ph.D.

Director

Exercise Physiology Division

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The skeleton's ability to withstand functional loadings without damage is primarily the result of the bones' capacity to remodel in response to the manner and magnitude of the loads imposed upon them. The consequence of this adaptive response is that skeletal mass and architecture are normally "tuned" to each individual's current mechanical load-carrying requirement. Under normal circumstances, in young, healthy individuals, this matching of structure and function results in adequate bone mass, appropriate architecture, and pain-free use.

7 The occurrence of stress fractures indicates a failure to maintain an adequate match between bones' structure and their functional requirement. Stress fractures occur both in well-trained athletes pushing for the limits of their performance, and in poorly-trained people, particularly women, following an abrupt change or increase in the level of their physical activity (1). The conventional view of the etiology of the "training up" type of stress fracture is that the remodelling processes are incapable of completing the necessary structural alterations before continued loading causes failure due to fatigue in the bone material. The

2

repair of this internal damage can only be accomplished by increased resorption, which aggravates the situation by further reducing the already inadequate effective bone area. At this stage, radiological evidence of a defect and/or an area of bone rarefaction may appear.

Treatment of the condition is relatively simple since reduction of strenuous activity allows the reparative remodelling process to run its course, following which the higher level of activity can be gradually reintroduced.

In order to investigate the etiology of stress fractures induced by abnormally high repetitions of cyclic loadings, we utilized the in vivo avian ulna preparation previously reported (2).

Intermittent loads from a modified Instron machine were applied to the ulna via its transfixing pins. The loads were adjusted to produce peak longitudinal strains at the midshaft of either 2000 or 3000 microstrain. The strain waveform was sinusoidal with a maximum loading and unloading strain rate of 50,000 microstrain/sec. Each bone was subjected to 30,000 strain cycles per day in a single period of loading. Loading was discontinued either when the animal showed discomfort or at 8 weeks,

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whichever was the sooner. The fewest number of cycles delivered to any bone was 30,000 (1 day), the largest was 1,600,000 cycles (54 days).

In previous experiments (2), using an identical preparation, 100 strain cycles per day, producing peak strains of 2000 microstrain at 10,000 microstrain/sec, had been demonstrated to be osteogenic. A similar response was elicited in this 30,000 cycle per day series. However, while transverse microradiographs of the ulna midshaft demonstrated the degree of intracortical remodelling in the 100 cpd series to be very low, in the 30,000 cpd series it was extensive. In addition, the large structural defects which were apparent in the bone cortices. These were not cracks or microdamage, but resorption spaces and expansion of vascular channels. Some of these channels extended from the periosteal surface endosteally and some completely traversed the cortex. These spaces, combined with the exuberant periosteal new bone formation with which they were associated, presented a similar appearance to naturally occurring stress fractures. The region of the most consistent and extensive intracortical remodelling was not that subjected to the greatest strain, but rather was located about the bone's neutral axis. Also, although enlarged vascular channels extended from the periosteal surface, the largest resorption cavities were in the center of the

cortex which is subjected to substantially lower strains than the periphery.

These data suggest that the most extensive and demonstrable bone defects associated with excessive repetitions of cyclic loadings are those resulting from resorption. The resorption of the cortex, and the eventual failure caused by the decrease in effective load bearing area, is thus more likely to be a consequence of the remodelling response to the prevailing strain situation than a failure of the material due to fatigue.

(1) Scully & Besterman (1982) Mil. Med. 147:285

(2) Rubin & Lanyon, Trans. O.R.S. 1983, p.70.

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